STUDIES ON EXTERNAL COUNTERPULSATION AS A POTENTIAL MEASURE FOR ACUTE LEFT HEART FAILURE

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In evaluation of measures which might be of help in massive myocardial infarction with shock, we have published work on left heart bypass and the physiologic and technical factors involved[1]. On embarkation on the broad study of support for this condition, it seemed to us wise also to consider all possible methods by which such support might be provided, and to seek not only the most effective one, but that which could be employed with a minimum of delay or manipulation.

Sarnoff et al. [2] clarified that the product of the pressure against which the left ventricle must eject blood into the aorta and the duration of that ejection, the time-tension index, is the chief determinant of the work of the heart, as measured by oxygen consumption. Subsequently Clauss et al. [3] undertook to remove blood from the aortic arch during ventricular ejection as a means of reduction of the pressure of ejection and thus of the work which the heart was required to perform. They returned this removed blood during that portion of the cardiac cycle during which the aortic valves were closed, so as to keep a satisfactory mean pressure for systemic perfusion. They have presented evidence which suggests a beneficial effect of this "counterpulsation" in certain types of cardiac damage. Soroff et al.[4] have done oxygen consumption studies in similar experiments and have demonstrated a reduction in oxygen utilization under these circumstances.

During the studies of some of us at the Thoracic Clinics of the Karolinska Institutet in the year 1960-61, an attempt was made to evaluate the possibility that such counterpulsation might be applicable without either arterial cannulation or heparinization. It was hoped that external compression of a sizable portion of the body, such as the hind quarter, when synchronized with the cardiac cycle, might bring about the same pressure changes in the aortic arch as those accomplished by Clauss and Soroff.

MATERIALS AND METHODS

Seventeen mongrel dogs were employed. These varied in weight from 5.8 to 18 Kg. All animals were anesthetized with intravenously administered barbiturate, usually inducing with 30 mg./Kg. of pentobarbital and adding additional barbiturate as indicated in the course of the experiments. In most animals a continuous, slow, intravenous infusion was maintained for satisfactory fluid balance. In addition the body temperature was recorded in most experiments and maintained in a normal range by means of electric heating pads. These experiments were divided into several categories.

In one animal the torso was divided at the level of the third lumbar vertebra with great care to maintain hemostatic control of the vessels of the caudal one-half. A cannula was fitted into the aorta, and the whole half-dog preparation mounted in an air-tight tank with the cannula projecting through to the outside. Efforts were made to determine the volume of blood which could be ejected from the hind quarters upon variation in the air pressure.

In a second series of experiments the left hind extremity was encased in a flexible double-layered polyethylene sleeve to permit application of external pressure to that extremity alone. Following heparinization, cannulas were placed in the proximal and distal ends of the divided left femoral artery just at the top of the encasing sleeve. These cannulas were connected through a flowmeter of the type described by Bruner[5]. Measurements of blood flow to the extremity were then correlated with applied external pressure.

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In a third series of experiments, a 7 Kg. dog was placed on a plywood board suspended so that the axis of rotation was horizontal and usually parallel with the long axis of the body, while the hind legs were at right angles to the long axis. The hind quarters were encased in a single sleeve made up of two layers of heavy, flexible, polyethylene sheeting between which air could be injected under desired pressure (Figure 1). A beaker of fluid was placed upon the animal board at a distance of 11 cm. from the axis of rotation. The volumes of fluid expressed from the hind quarters by application of external pressure or permitted to return upon release of that pressure were roughly estimated on the basis of changes in beaker fluid content needed to restore balance. In these experiments an intratracheal tube was routinely placed. The intravenous infusion tubing, a tube used to alter the fluid content of the beaker, the air pressure line to the external compression sleeve, and the electric cord of a telethermometer were brought on to the balanced preparation through the axis of rotation.

In a fourth series of experiments, an effort was made to provide counterpulsation approximately sychronously with the spontaneous heart beat. For this purpose the double-sleeve encasement of the hind quarters was again employed, but the sleeve was filled with tap water at body temperature and was in turn encased in a rigid wooden box, which was provided with an aperture at one end precisely to fit the contour of the dog just above the pelvis. Counterpulsation in this preparation was provided with a false top to the box, which in turn was activated manually by a lever two meters in length. The pressures in the sleeve and in the aortic arch were recorded on an Elema Mingograf recorder.

Finally, a preparation for external counterpulsation with a similar rigid box was constructed in the laboratories in Brooklyn, utilizing the same principles as those employed in Stockholm, but increasing the sleeve pressure by means of a 10-14 lb. /in^2. pressure line with an air reservoir tank and a three-quarter inch solenoid communicating through a one-inch iron pipe connection to a heavy rubber bladder in the bottom of the encasing box. The solenoid is activated through an electronic circuit so as to provide external pressure of any desired duration, pressure level, or lag after initiation of ventricular contraction. In some experiments, the electronic circuit was triggered by the R wave of the QRS complex of the electrocardiograph, and in others by the beginning rise in left ventricular pressure.

In the evaluation of this apparatus, a tracheal tube has routinely been placed for utilization of a Jefferson respirator, and the left chest has been opened for direct placement of polyethylene catheters (0.054" in external diameter) into the left ventricle and into the aorta. In most experiments, the electrocardiograph and pressures in the left ventricle, the aortic arch, and the compression sleeve have been recorded on a four-channel Sanborn recorder. In some experiments, intra-abdominal pressure and venous pressure have been recorded instead of some of the above.

RESULTS

Attempts to express blood from the arterial tree of the amputated caudal one-half dog preparation were fruitless. This was true regardless of the position of the compression chamber with regard to gravity.

Total occlusion of the femoral arterial tree was accomplished by a pressure sleeve ensheathing the hind leg with pressures little higher than the systolic arterial blood pressure. The release of such ensheathing pressure within a period of three seconds was followed in the ensuing four seconds, the time required for flow measurement, by blood flow rates higher than the controls (Figure 2). Following short periods of compression occlusion, this acceleration was less marked than following occlusion of several minutes.

In efforts to quantify the sleeve expression of fluid from the venous and lymphatic systems, on the one hand, and from the arterial tree, on the other, the data suggest that large volumes are expressed by pressures below 50 mm. Hg (Figure 3). The volumes expressed above this pressure are in the range of 3 ml. /Kg. of body weight. Abrupt rises from 0 to 60 mm. Hg sleeve pressure in this same preparation expelled in eight observations sufficient fluid to be counterbalanced by a mean of 11 ml. /Kg. of body weight. Abrupt rises from 0 to 160 mm. Hg expelled in four observations a mean of enough fluid to be counterbalanced by 14.5 ml. of water per Kg. of body weight.

Attempts manually to synchronize the sleeve pressure with the heart beat of the dog were too inconsistent for reliable interpretation, and no left ventricular tracings were undertaken. Nonetheless, the shape of the aortic pressure tracing was successfully altered to indicate a lowered left ventricular ejection peak pressure, with an elevation during ventricular diastole to higher levels than the control systolic pressure. Such counterpulsation was noted to cause wide changes in the baseline and voltage of the electrocardiograph.

The electronically controlled external counterpulsator unit has proven capable of raising the sleeve pressure to 150 mm. Hg in 0.04 sec. and of permitting it to drop to the atmospheric level in 0.06 sec. It has proven possible to lower the peak aortic pressure 5 to 10 mm. Hg, to raise the aortic pressure during left ventricular diastole to levels higher than the control systolic pressure, and to lower the time-tension index 8 to 10%. Variations in the electrocardiograph, especially after coronary artery ligation, have caused us to evaluate the principle of external counterpulsation using the rise in left ventricular pressure instead of the R wave to trigger our system.

There is some variation in factors among dogs, but, as an example, good changes in pressure tracings in an 11 Kg. dog with pulse rate of 111 were obtained with 10 lb./in.^{2} air pressure, a lag after the R wave of 0.12 sec., and a duration of compression of 0.32 sec. A representative experiment is shown in Figure 4. Some reduction in time-tension index was commonly seen even though the sleeve pressure peak was less than the systolic blood pressure. No oxygen utilization studies have as yet been performed.

Although no precise data serve to document rapid cardiac deterioration when improper synchronization of external compression is employed, nonetheless the definite impression was gained that the heart tolerated very poorly external counterpulsation which was poorly timed.

A small number of observations on the effect of external counterpulsation as here utilized upon intra-abdominal pressure and upon central venous pressure indicated that the effects on these two values were very small. Further studies are in progress.

DISCUSSION

These studies demonstrate that sufficient blood can be expressed from the buttocks and hind legs of the dog to be effective in lessening the time-tension index and the peak left ventricular pressure, while providing a peak aortic pressure during left ventricular diastole equal to the control systemic blood pressure.

The possible effect of such counterpulsation upon the venous return to the heart is still an unresolved question. Whether there is a hazard of augmentation of venous return and secondarily of pulmonary edema must be clarified before clinical application.

The mechanical problems of rapid external pressure change appear to be readily soluble. In the present studies we have avoided the problems of poor emptying of the arterial tree which arise from gravitational factors by the use of the horizontal position. We have satisfied ourselves that the external encasement must have rigid limits and that the content which transmits pressure to the tissues must be incompressible. The use of air for accomplishment of pressure changes was dictated by the need for rapid exchanges, but the compressibility of air has dictated utilization of volumes as small as possible and of conduits as large and short as feasible. We are also convinced by the study on the amputated half-dog that air pressure on a fluid-filled flexible tube, such as a normal artery, will express the content only with and not against gravity. It is equally axiomatic that a hyperbaric solution must be employed if the extremities are to be in a dependent position.

Although we have experienced real difficulty in triggering our pressure system by the R wave of the electrocardiograph, the occasional experiment in which this trouble was totally absent suggests this to be a soluble problem. It is clear that left ventricular pressure and aortic pressure cannot easily be employed clinically for triggering. With all of our efforts, many of our experiments have been failures and have not shown either reduction of left ventricular pressure or of time-tension index. It appears likely that the complexity of the apparatus and of the experimental preparation rather than a fault in the thesis has been our greatest obstacle. Whether such obstacles will prove of sufficient magnitude to preclude clinical application at a later time, only further study can resolve.

It is possible that the diffuse vascular disease which exists in most patients with myocardial infarction may limit the application of external counterpulsation. Only by studies on the expressible volume in human subjects can this question be resolved. Such studies are in progress.

CONCLUSIONS

External sleeve compression of the lower extremities of the dog to levels very little above the arterial blood pressure can stop completely the arterial flow in them. The arterial blood expressible is of the order of magnitude of 3 ml./Kg. of body weight.

At pressures below 50 mm. Hg, much larger extremity weight losses occur. These have been attributed to expression of venous blood and lymph.

The technical problems of external counterpulsation have been resolved sufficiently to demonstrate that the time-tensionindex can be reduced, that the left ventricular pressure can be lowered, and that a peak of aortic blood pressure as high as the control systolic blood pressure can be produced while the aortic valve is closed.

ACKNOWLEDGEMENT AND FOOTNOTE

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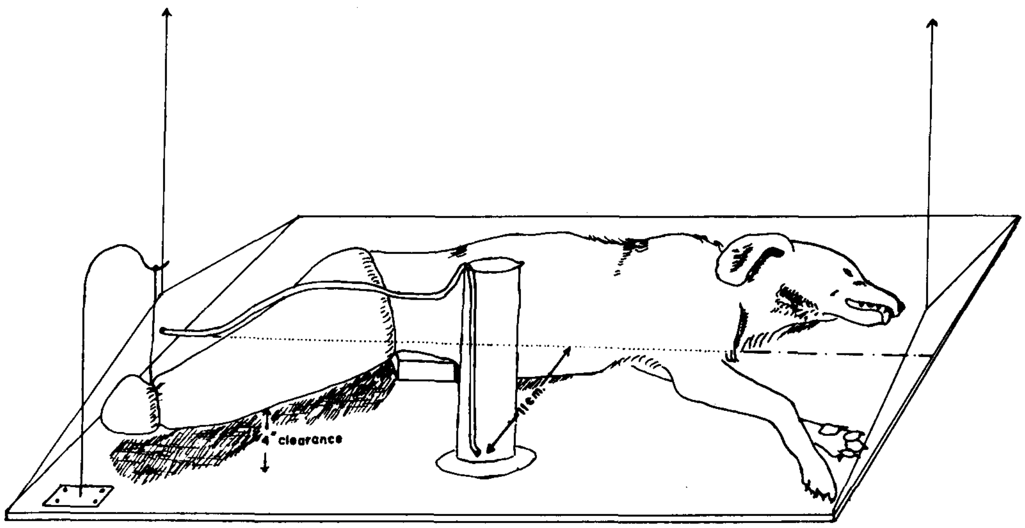

Figure 1. Pattern of balance experiments to estimate roughly the volumes of fluid expressible from the hind quarters by double-sleeve external compression. See text for details. All connections except the water tube are omitted for clarity of illustration.

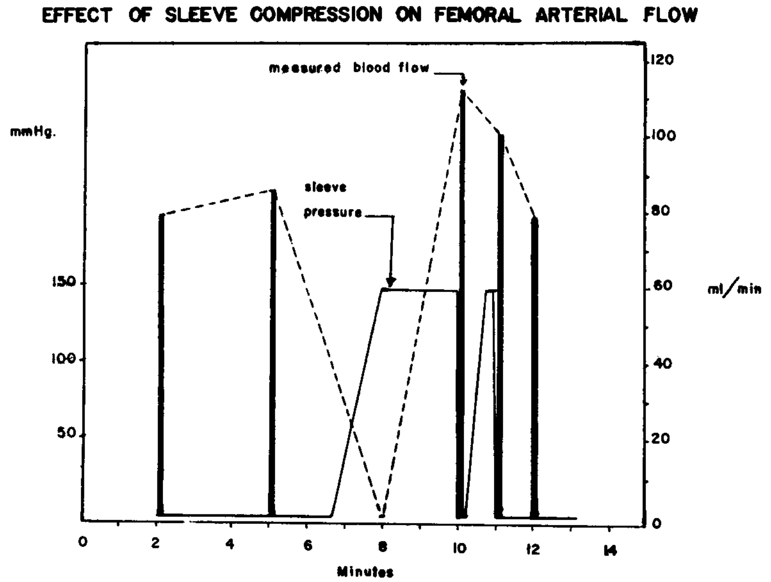

Figure 2. Femoral arterial blood flow during external sleeve compression in a 5.8 Kg. puppy with a mean blood pressure of 85 mm. Hg. The solid bars indicate blood flow determinations made with a stopwatch and bubble flowmeter as outlined by Bruner.

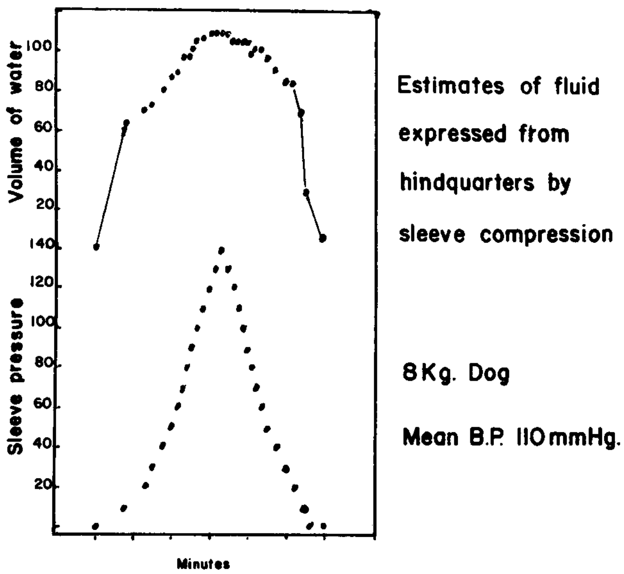

Figure 3. Estimates of fluid expressed from the canine hind quarters by sleeve compression in an 8 Kg. subject with mean blood pressure of 110 mm.Hg. at the start.

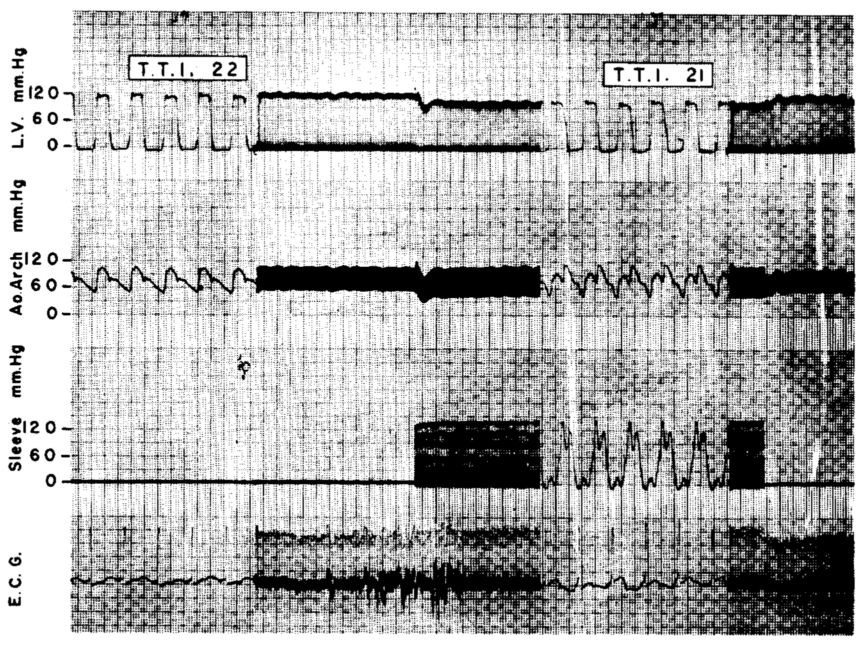

Figure 4. Changes in left ventricular and aortic pulse wave with external counterpulsation applied to the hind quarters. The left ventricular component of the aortic pressure curve is reduced from 112 mm.Hg to 90 mm. Hg. The left ventricular peak pressure is lowered from 112 mm.Hg to 100 mm.Hg. The imposed aortic peak pressure is 114 mm.Hg. Paper speeds: slow-0.25 mm./sec.; fast-25 mm./sec. Use of a slow paper speed permits perspective as to relatively slow responses. For 5 to 6 sec. there was a rise in left ventricular and aortic arch pressure, followed by a 35-mm. Hg drop, with resumption in about 30 sec. of a peak pressure in diastole slightly greater than the control systolic pressure. The left ventricular pressure rose late in the first 30 sec. of external counterpulsation to a level 12 to 15 mm.Hg below the control left ventricular systolic pressure. At the end of periods of external counterpulsation, the changes in left ventricular pressure were essentially mirror images of those at the beginning. The aortic arch pressure changes were slower to revert to control levels, but did so in 1-1/2 min.

DR. SALISBURY: This interesting paper reproduces in a different way Mr. Birtwell's counterpulsation. Unfortunately, Dr. Dennis did not really create failing hearts, and the data do not tell how this technique will alter the left ventricular diastolic pressure. It is now believed that high left ventricular diastolic pressure is the only reliable criterion from which the onset of pulmonary edema can be predicted. A fall of high left ventricular diastolic pressure is therefore the only criterion which indicates the usefulness of any therapy in acute left ventricular failure. The oxygen consumption of the heart, the area under the pressure curve, or any other parameters do not indicate the presence or absence of left ventricular failure. Unless you can show significant changes in the left ventricular diastolic pressure, we really cannot know whether your treatment is useful.

DR. DENNIS: I think you're giving me a carbon copy of my final paragraph, Peter. I quite agree it isn't not yet resolved at all, and this is the reason for the conservatism in expressing this.

We have done one or two studies on coronary occlusion in association with this, but we're not prepared to say anything about it as yet.